REVIEWS

Overview on eating disorders

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Summary. There is a commonly held view that eating disorders are lifestyle choice. Eating disorders are actually serious and often fatal illnesses, obsessions with food, body weight, and shape may also signal an eating disorders. Common eating disorders include anorexia nervosa, bulimia nervosa, night-eating syndrome, eating disorders not otherwise specified and binge-eating disorders. Eating disorders occur in men and women, young and old, rich and poor and from all cultural backgrounds; they result in about 7000 death a year as of 2010, making them the mental illnesses with the highest mortality rate. The chance for recovery increases the earlier they are detected, therefore, it is important to be aware of some of the warning signs of an eating disorder. In this review, different types of eating disorder, their side effects, complications and treatments are discussed.

Key words: eating disorders, types, side effects, complications, treatments

Introduction

Whether it is the effect of the media, family or friends, the number of eating disorders has significantly increased and they are becoming more and more prevalent. There are five classifications of eating disorders: anorexia, bulimia, binge eating disorder (BED), eating disorders not otherwise specified (EDNOS) and night eating syndrome (1). Over seven million girls and women and one million boys and men will suffer from an eating disorder in their lifetime. Up to 3.7% of females will be diagnosed with anorexia nervosa and an estimated 4.2% will have bulimia nervosa (2). The majority of adolescent patients seen in referral centers fit into a third category (EDNOS) and does not fit strict criteria for either anorexia or bulimia (3). Nineteen percent of college-aged females are bulimic; many go undiagnosed until much later. At the other end of the spectrum, 1% to 5% of the population falls into the category of binge eating disorder, not yet an approved psychiatric diagnosis (4). Anorexics are more likely to be female (90%-95%); 80% of bulimics are female and 60% of BEDs are female (5). Eating disorders begin early, with 10% being diagnosed in children less than 10 years of age. One third of patients are diagnosed as preteens and adolescents up to age 15. In total, 86% of patients are diagnosed with eating disorders before the age of 20 (6).

Etiology of Eating Disorders

The eating disorders have traditionally been viewed as sociocultural in origin. However, recently it was found that genetics tend to have a strong influence on these disorders (7). Current research demonstrates that eating disorder symptoms may be as common or more common among certain ethnic groups (Asians, blacks, and Hispanics) when compared with whites (8). There was no difference found in dieting and restraint scores between Asian, Latino, and white adolescent girls and boys (9) and no difference in binging or BED in obese patients who sought to lose weight with bariatric surgery (10). However, an analysis of 18 studies (1987-2001) concluded that African-American women were less likely than white women to have an eating disorder (11). As well, a study in school age girls dem-

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onstrated that Native American girls had higher rates of restricting/purging and dieting than white or nonwhite/non-Native American populations (12).

Types of Eating Disorders

1. Anorexia nervosa (AN)

Anorexia nervosa is a highly distinctive serious mental disorder. It can affect individuals of all ages, sexes, sexual orientations, races, and ethnic origins; however, adolescent girls and young adult women are particularly at risk (13, 14). The disorder involves the fear of gaining weight, having a distorted body image, a refusal to maintain normal weight, and the use of extreme measures to keep the weight off. Anorexia is typically diagnosed after a person is 25–30 percent below the normal weight for three months or more (15). Additionally, cognitive and emotional functioning are markedly disturbed in people with this disorder (16).

Typically, two sub-types of anorexia are identified. First, restricting-type anorexics (R-AN) lose weight purely by dieting and exercising without binge eating or purging. Second, binge-eating/purging-type anorexics (BP-AN) also restrict their food intake and exercise to lose weight, but periodically engage in binge eating and/or purging (17).

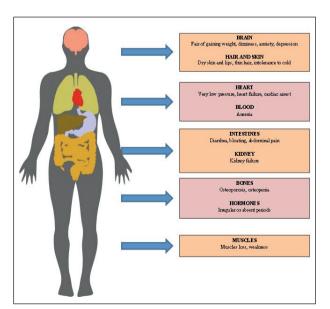


Figure 1. Physical signs and effects of anorexia nervosa

Anorexia is often associated with denial of illness and resistance to treatment. Consequently it is difficult to engage individuals with AN in treatment, including nutritional restoration, and weight normalization (18). The physical signs and effects of anorexia are presented in figure 1.

2. Bulimia nervosa (BN)

Bulimia nervosa is a serious, potentially life-threatening eating disorder. It is characterized by a cycle of bingeing and compensatory behaviors such as self-induced vomiting designed to undo or compensate for the effects of binge eating (19). Patients diagnosed with bulimia nervosa follow closely with patients diagnosed with binge-purge anorexia (1). Bulimia is diagnosed if the binge-purge cycle occurs at least two times a week. The act of purging can cause severe damage to the esophagus and teeth and it can also cause the gag reflex to be less sensitive (1).

Non-Purging type of bulimia is also diagnosed and is characterized by using other inappropriate methods of compensation for binge episodes, such as excessive exercising or fasting. In these cases, the typical forms of purging, such as self-induced vomiting, are not regularly utilized (20). The physical signs and effects of bulimia nervosa are presented in figure 2.

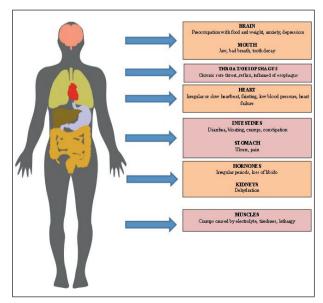


Figure 2. Physical signs and effects of bulimia nervosa

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3. Binge-eating disorders (BED)

According to the Diagnostic and Statistical Manual of Mental Disorders (DSM), 5th edition, binge-eating disorder is defined by several criteria (21). Individuals must report consuming an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances in addition to experiencing a loss of control over one's eating behavior during this time (21). In addition, at least three of the following characteristics must also be present: summing food much more rapidly than normal; eating food until uncomfortably full; consuming large amounts of food when not feeling physically hungry; consuming food alone to avoid embarrassment; or feeling disgusted, depressed, or guilty after the eating event (22). The diagnosis also requires that a significant amount of distress be associated with the binge episodes, which must occur at least once per week for 3 months or more. Lastly, the disorder must not be accompanied by any regular compensatory behavior, nor should the binge eating occur solely during an episode of bulimia nervosa or anorexia nervosa (22). The physical signs and effects of binge-eating disorder are presented in figure 3.

4. Eating disorders not otherwise specified (EDNOS)

Eating disorders not otherwise specified is much used by clinicians yet largely ignored by researchers. It

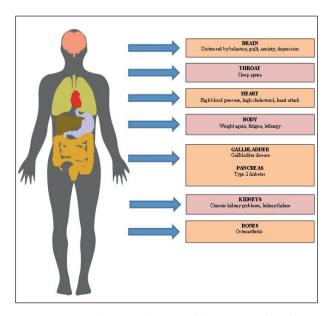


Figure 3. Physical signs and effects of binge eating disorder

is the category for disorders that do not meet the criteria for any other specific eating disorder and accounts for about 50% of eating disorders (23, 24). Although patients with EDNOS do not meet the diagnostic criteria for either AN or BN, if the disordered behaviors continue, they may progress to frank AN or BN. For example, some patients may have met all criteria for AN with the exception of missing three consecutive menstrual periods, or they may be of normal weight and purge without bingeing. Although patients may not present with medical concerns and treatment modality depend on the severity of impairment and the symptoms (24).

Fairburn and Bohn described two subtypes as particularly common for EDNOS. The first are instances where the individual's presentation closely resembles AN or BN nervosa, but he or she just fails to meet the diagnostic thresholds. The second subtype are cases in which the clinical features of AN and BN are combined in ways other than in the two recognized syndromes (25).

5. Night-eating syndrome (NES)

The other prominent form of disordered eating related to overweight and obesity is NES. NES was first described by Stunkard et al. among a group of individuals with obesity seeking weight loss treatment (26). They reported that those with the syndrome consumed a large majority of their caloric intake (25% or more) at a time when individuals without obesity would not be eating. In addition, the patients experienced insomnia and morning anorexia. Attention to NES was neglected until the late 1990's, when the focus of eating-related research shifted in response to the growing prevalence of obesity in the United States (27). Since this time, the definition of NES has varied. For example, in later years, Stunkard's definition was expanded to include nocturnal ingestions (28).

NES is characterized by recurrent episodes of night eating, which is described as either excessive food consumption in the evening (after dinnertime, i.e., evening hyperphagia) or eating after awakening from sleep (i.e., nocturnal ingestions). NES is also characterized by at least three of the following symptoms: morning anorexia, the presence of a strong urge to eat between dinner and sleep and/or during

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the night, sleep onset and/or maintenance insomnia, frequently depressed mood or mood worsening in the evening, and a belief that one cannot get back to sleep without eating (28, 29). In order to be diagnosed with NES, individuals must be aware of and be able to recall the eating episodes. These symptoms must also cause significant distress and/or impairment in functioning and not be better explained by external factors or another disorder, such as a sleeping disorder or other disordered eating pattern (30). NES is classified in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM5) as an "other specified feeding or eating disorder."

Side effects of eating disorders

1. Osteoporosis

Anorexics are at increased risk of osteoporosis due to lowered intake, being under- weight, and decreased estrogen related to amenorrhea. Calcium supplementation in pubertal girls may increase peak bone mass (31). Calcium supplementation may increase the beneficial effects of physical activity on bone (32). Deficiency of vitamin D in young people can affect their ability to reach peak bone mass (33). Special risks in eating disorder patients for osteoporosis include the following:

- Anorexic girls (aged 13-23 years) who also suffer from depression may be at higher risk for osteoporosis than those without depression; the reason for this finding is not known (34).
- . Amenorrhea in anorexic women and young girls may indicate the onset of estrogen deficiency, which can have a negative effect on bone density and peak bone mass.
- Under-nutrition can affect bone density through deficiency of anabolic hormones such as insulin like growth factor I; in addition, low weight is also a risk factor for lowered bone mass (35).
- Data indicate that osteoporosis could be considered a risk factor for periodontal disease progression, especially in subjects with preexisting periodontitis (36).

2. Taste receptors damaged

For all four taste stimuli (sweet, salty, sour, and bitter), intensities on the palate have been found to be lower in bulimic subject than in control subject (37), reduced taste sensitivity affected only the palate and not the whole mouth. Specifically, taste receptors located on the palate may become damaged because vomit is directed toward the roof of the mouth during purging (37, 38).

3. Oral Health

The association between oral pathology and eating disorders is most clearly established in cases with frequent self-induced vomiting, regardless of whether the diagnosis is anorexia or bulimia, and is characterized by dental erosion on palatal surfaces. Dental caries and dry mouth secondary to salivary gland dysfunction also occur (39). Gingival inflammatory changes due to vitamin C deficiency/scurvy are also observed (40).

4. Others

There were other side effects as well, including decreased concentration and other cognitive changes; physical changes that included decreased need for sleep; gastrointestinal problems; dizziness; headaches; noise and light sensitivity; weakness; fluid retention; cold intolerance; and difficulties with hearing and sight. There was a 40% slowing of basal metabolic rate, low body temperature, decrease in heart rate, and respiration (41).

Complications of eating disorders

No rehabilitation works 100% every time and a risk of relapse is always present. Re-feeding is one of the most prevalent complications characterized by the inability of the body to cope with the extreme change in metabolic function. The main signs of the re-feeding syndrome are (42):

- Hypophosphatemia, hypokalemia and hypomagnesaemia
- · Heart failure
- Salt and water retention
- Depletion of vitamins such as B1, B6

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These symptoms are caused by the change in metabolism in the body from fat to carbohydrates. When an anorexic patient starves themselves they are using stored fat as the primary source of energy. But when they start eating again their bodies can switch from using stored fat as energy to using carbohydrates from food again. This change will lead to insulin being released from the pancreas to aid in the uptake of glucose. When the insulin is released cells will start to increase the amount of glucose, phosphate, potassium, magnesium and water that they take in (43).

To avoid re-feeding syndrome, levels of phosphorus, magnesium, potassium and calcium should be determined for the first 5 days and every other day for several weeks; electrocardiogram should be also performed (18). If indicated, during the first days of refeeding, large amounts of multi-vitamins and minerals, in particular potassium, thiamine, phosphate and magnesium, should be provided. Again, strict monitoring is needed to prevent vitamin A and D toxicity in case of excessive supplements (44).

Types of treatment on eating disorders

1. Pharmacological treatments

Medications are generally useful for patients with bulimia nervosa and BED. Common forms of pharmacotherapy include antidepressants, antiepileptic medications, anti-obesity, and stimulant medications (45). For bulimia nervosa, antidepressant medications are the primary pharmacologic treatment (46). The evidence for the use of fluoxetine in the treatment of bulimia nervosa comes in the form of various case reports, systematic studies, and double-blind, randomized placebo controlled trials (47). Tricyclic antidepressants and monoamine oxidase inhibitors are also found to be effective were also found to be more effective than placebo in decreasing the binging and vomiting in patients with bulimia nervosa (48). Ondansetron at 24mg/day is also reported to reduce binge eating and self-induced vomiting in a small placebo-controlled study of 29 patients with bulimia nervosa (49).

For BED, lisdexamfetamine is reported to be generally well tolerated and effective, and is the first medication to be indicated by the FDA for treatment of BED (46). The anticonvulsant topiramate administered at a dose of 25 to 600mg daily is found to significantly reduce binge frequency and weight (50).

For anorexia nervosa, there is limited evidence supporting benefits of medications and different treatments were used for treating the accompanying symptoms. Olanzapine appears to demonstrate some benefit for weight gain and transdermal administration of hormonal agents is also being explored for improving bone health in anorexia nervosa (46).

2. Family-based treatment (FBT)

Although early models of family therapy for AN focused on addressing problematic aspects of the family that were believed to contribute to the development and maintenance of AN (51, 52), more recent models have focused on reducing blame and utilizing the family as a resource for recovery (53). In FBT, parents play a central role in restoring their child's health, and siblings are encouraged to provide emotional support to their ill sibling. The FBT should happen in the home during parental meal and needs the support of both parents. If parents do not have a shared understanding of how to undertake these tasks, they may unintentionally undermine each other (54).

3. Inpatient

Inpatient treatment is usually for very seriously ill patient who are usually the ones with cardiac or severe psychological issues that might need special medical attention throughout their treatment. These patients are fed by nasogastric feeding in order to reduce the risk of re-feeding syndrome and insulin spikes that can cause serious problems (55). Patients have also shown that they have less abdominal distention, nausea, and bloating (55). By being fed this way, doctors are able to add in more necessary fat to the diet without the patient objecting leading to a decreased hospital stay.

4. Outpatient

The standard nutritional treatment for outpatient is progressive bolus oral feeding (55). This is when the patient has a nutritionist set up a plan for what they need to eat to meet their goal caloric intake as well as nutritional needs. But some patients have had digestive issues such as nausea, bloating and pain from re-

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turning to normal eating too quickly (55). The biggest consequence of this form of treatment is that it can lead to re-feeding syndrome and refusal to eat altogether. Many patients will struggle with the idea of eating solid food again especially enough to meet the caloric intake goal needed to make them healthy.

Conclusion

Eating disorders affect not only the diagnosed patients, but the families surrounding them. They can be triggered by society trends, genetics, and family and can develop during any stage in life, classified as a medical illness. Although these conditions are treatable, the symptoms and consequences can be detrimental and deadly if not addressed. They commonly coexist with other condition, such as anxiety disorder, substance abuse, or depression. Eating disorders can lead to heart and kidney problems and even death. Treatment involves monitoring, talk therapy, nutritional counseling, and sometimes medicines. People with eating disorder suffer of osteoarthritis, Kidney failure, high blood pressure, diarrhea, dizziness, etc. There are many complications that can arise with treatment such as refeeding syndrome and hypophosphatemia, which can lead to patient distress or fatality. Eating disorders are a lifelong battle even after treatment is completed.

References

- Weiten W. Psychology themes & variations. 8th Ed. Las Vegas, NV: University of Nevada W. 2011, 570p.
- 2. American Psychiatric Association Work Group on Eating Disorders. Practice guideline for the treatment of patients with eating disorders (revision). Amer J Psych. 2000; 157(Suppl. 1): 1-39.
- Merikangas KR, He J, Burstein M, et al. Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Study-Adolescent Supplement (NCS-A). J Am Acad Child Adolesc Psychiatry. 2010; 49(10): 980-989.
- 4. Smink FRE, van Hoeken D, Hoek HW. Epidemiology of Eating Disorders: Incidence, Prevalence and Mortality Rates. Curr Psychiatry Rep. 2012; 14(4): 406–414.
- Wade T, Keski-Rahkonen A, Hudson J. Epidemiology of eating disorders. In: Tsuang M, Tohen M. Textbook in Psychiatric Epidemiology (3rd ed). New York: Wiley, 2011, 660p.
- 6. Stice E, Marti CN, Shaw H, Jaconis M. An 8-year longitudinal study of the natural history of threshold, sub-threshold,

- and partial eating disorders from a community sample of adolescents. Journal of Abnormal Psychology. 2010; 118(3): 587-97.
- 7. Wade B. The Genetics of Eating Disorders. Psychiatry (Edgmont). 2004; 1(3): 18-25.
- 8. Shaw H, Ramirez L, Trost A, et al. Body image and eating disturbances across ethnic groups: More similarities than differences. Psych of Addic Behav. 2004; (18): 12-18.
- Cachelin FM, Weiss JW, Garbanati JA. Dieting and its relationship to smoking, acculturation, and family environment in Asian and Hispanic adolescents. Eating Disorders. 2004; (11): 51-61.
- Sanchez-Johnson L, Dymek M, Alverdy J, Le Grange D. Binge eating and eating-related cognitions and behavior in ethnically diverse obese women. Obesity Research. 2003; (11): 1002-1009.
- O'Neill SK. African American women and eating disturbances: A meta-analysis. Journal of Black Psychology. 2003; (29): 3-16.
- 12. Lynch WC, Eppers KD, Sherrod JR. Eating attitudes of Native American and white female adolescents: a comparison of BMI-and age-matched groups. Ethnicity & Health. 2004; (9): 253-266.
- 13. Zipfel S, Mack I, Baur LA, et al. Impact of exercise on energy metabolism in anorexia nervosa. J Eat Disord 2013; (1): 37.
- 14. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. Washington, DC: American Psychiatric Association. 2013, 947p.
- 15. American Dietetic. Position of the American dietetic association: Nutrition intervention in the treatment of anorexia nervosa, bulimia nervosa, and other eating disorders. Journal of the American Dietetic Association. 2007; 106(12): 2073-82.
- Zipfel S, Giel K, Bulik C, Hay P, Schmidt U. Anorexia nervosa: aetiology, assessment, and treatment. Lancet psychiatry, 2015; 2(12): 1099-111.
- 17. Kelly AC, Carter JC. Eating disorder subtypes differ in their rates of psychosocial improvement over treatment. J Eat Disord. 2014; 2: 2.
- 18. American Psychiatric Association: Treatment of patients with eating disorders, 3rd ed. Am J Psychiatry. 2006; 163: 4-54.
- Favaro A, Caregaro L, Tenconi E, Bosello R, Santonastaso P. Time trends in age at onset of anorexia nervosa and bulimia nervosa. Journal of Clinical Psychiatry. 2009; 70(12): 1715-21.
- 20. Ramacciotti CE, Coli E, Paoli R, et al. The relationship between binge eating disorder and non-purging bulimia nervosa. Eat Weight Disord. 2005; 10(1): 8-12.
- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. DSM-5. Washington DC. 2013, 991p.
- 22. Stice E, Bohon C. Eating Disorders. In: Child and Adolescent Psychopathology, 2nd Edition, Beauchaine Th, Linshaw S, 1st ed. New York: Wiley, 2012, 912.
- 23. Martha M, Levine P, Levine RL. Psychiatric Medication. In: Treatment of Eating Disorders, 2010.
- 24. Spear BA. Eating Disorders. In: Handbook of Clinical Nutrition, (4 Ed), 2006.

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- 25. Fairburn CG, Bohn K. Eating disorder NOS (EDNOS): an example of the troublesome "not otherwise specified" (NOS) category in DSM-IV. Behav Res Ther. 2005; 43(6): 691–701.
- Stunkard J, Grace WJ, Wolff HG. The night eating syndrome: a pattern of food intake among certain obese patients. Am. J. Med; 1955 (19): 78–86.
- 27. Birketvedt GS, Florholmen J, Sundsfjord J, et al. Behavioral and neuroendocrine characteristics of the night-eating syndrome. JAMA. 1999; 282(7): 657-63.
- Allison C, Lundgren D, O'reardon J, et al. Proposed diagnostic criteria for night eating syndrome. Int. J. Eat. Disord. 2010; (43): 241-247.
- 29. De Zwaan M, Marschollek M, Allison KC. The night eating syndrome (NES) in bariatric surgery patients. Eur. Eat. Disord. Rev. 2015; (23): 426-434.
- 30. Howell MJ, Schenck CH, Crow SJ. A review of nighttime eating disorders. Sleep Med Rev. 2009; 13(1): 23-34.
- 31. Dodiuk-Gad RP, Rozen GS, Rennert G, et al. Sustained effect of short-term calcium supplementation on bone mass in adoles- cent girls with low calcium intake. Am J Clin Nutr. 2005; (81): 175-188.
- Hemayattalab R. Effects of physical training and calcium intake on bone mineral density of students with mental retardation. Research in Developmental Disabilities. 2010; 31: 784–789.
- 33. Calvo MS, Whiting SJ, Barton CN. Vitamin D intake: a global perspective of current status. J Nutr. 2005; (135): 310-316.
- 34. Konstantynowicz J, Kadziela-Olech H, Kaczmarski M, et al. J Clin Endocrinol Metab, 2005; (90): 5382-5.
- 35. Miller KK. Mechanisms by which nutritional disorders cause reduced bone mass in adults. J Womens Health (Larchmt). 2003; (12): 145-150.
- 36. Esfahanian V, Shamami MS, Shamami MS. Relationship between Osteoporosis and Periodontal Disease: Review of the Literature. J Dent (Tehran). 2012; 9(4): 256–264.
- 37. Garcia-Burgos D, Maglieri S, Vögele C, Munsch S. How Does Food Taste in Anorexia and Bulimia Nervosa? A Protocol for a Quasi-Experimental, Cross-Sectional Design to Investigate Taste Aversion or Increased Hedonic Valence of Food in Eating Disorders. Front Psychol. 2018; 9: 264.
- 38. Grabenhorst F, Rolls ET, Bilderbeck A. How cognition modulates affective responses to taste and flavor: top-down influences on the orbitofrontal and pregenual cingulate cortices. Cereb. Cortex. 2008; 18, 1549–1559.
- Frydrych AM, Davies GR, McDermott BM. Eating disorders and oral health: a review of the literature. Aust Dental J. 2005; 50: 6-15.
- Omori K, Hanayama Y, Naruishi K, et al. Overgrowth caused by vitamin C deficiency associated with metabolic syndrome and severe periodontal infection: a case report. Clin Case Rep. 2014; 2(6): 286-95.
- 41. Latner JD, Wilson GT. Binge eating and satiety in bulimia nervosa and binge eating disorder: effects of macronutrient intake. Int J Eat Disord. 2004; 36(4): 402–15.

- 42. Gentile G, Lessa C, Cattaneo M. Metabolic and nutritional needs to normalize body mass index by doubling the admission body weight in severe anorexia nervosa. Clinical Medicine Insights: Case Reports. 2013; (6): 51-56.
- 43. Crook A, Hally V, Panteli V. The importance of the re-feeding syndrome. Nutrition. 2001; 17(78): 632-637.
- 44. NICE: Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders (Clinical Guideline 9). London: National Collaborating Centre for Medical Health; 2004.
- Reas L, Grilo M. Pharmacological treatment of binge eating disorder: up to date review and synthesis. Expert Opin. Pharmacother. 2015; (16): 1463-1478.
- Davis H, Attia E. Pharmacotherapy of eating disorders. Curr Opin Psychiatry. 2017; 30(6): 452-457.
- Zhu AJ, Walsh BT. Pharmacologic treatment of eating disorders. Can J Psychiatry 2002; 47: 227-34.
- Bacaltchuk J, Hay P. Antidepressants versus placebo for people with bulimia nervosa. Cochrane Database Syst Rev. 2003;
 CD003391.
- 49. Faris PL, Kim SW, Meller WH, et al. Effect of decreasing afferent vagal activity with ondansetron on symptoms of bulimia nervosa: a randomised, double-blind trial. Lancet. 2000; 355(9206): 792-7.
- 50. McElroy SL, Arnold LM, Shapira NA, et al. Topiramate in the treatment of binge eating disorder associated with obesity: a randomized, placebo-controlled trial. Am J Psychiatry. 2003; 160(2): 255-61.
- Minuchin S, Rosman L, Baker L. Psychosomatic families: Anorexia nervosa in context. Cambridge, MA: Harvard University Press. 1978, 351p.
- 52. Stanton M, Welsh R. Systemic Thinking in Couple and Family Psychology Research and Practice. Couple and Family Psychology: Research and Practice 2012; 1 (1): 14 –30.
- 53. Eisler I, Dodge L, Wallis A. What's new is old and what's old is new: The origins and evolution of eating disorders family therapy. In: Loeb KL, Le Grange D, Lock J. Family therapy for adolescent eating and weight disorders: New applications New York, NY: Routledge/Taylor & Francis. 2015, 454.
- 54. Dimitropoulos G, Freeman E, Lock J, Le Grange D. Clinician perspective on parental empowerment in family-based treatment for adolescent anorexia nervosa. Journal of Family Therapy. 2017; 39(4): 537-562.
- 55. Agostino H, Erdstein J, Di Meglio G. Shifting paradigms: Continuous nasogastric feeding with high caloric intakes in anorexia nervosa. Journal of Adolescent Health. 2013; 53(5): 590-594.

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